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Nafamostat mesilate, a synthetic protease inhibitor, attenuated hypercoagulability in a canine model of hemorrhagic shock.

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Hypercoagulability is known to occur in the early phase of hemorrhagic shock. The prolongation of excessive clot formation after recovery from a shock state leads to the formation of microthrombi or disseminated intravascular coagulation which disturbs microcirculation, damaging organ function. The aim of the present study is to investigate the beneficial effect of a synthetic protease inhibitor, 6-amidino-2-naphthyl p-guanidinobenzoate dimethanesulfonate (nafamostat mesilate), in the attenuation of hypercoagulability in hemorrhagic shock. A model of hemorrhagic shock that simulates the clinical course of injured patients was created in anesthetized dogs. The animals were divided into two groups: a control group (group-C, n = 9) and an experimental group (group-E, n = 9). Animals received saline or 0.2 mg/kg of nafamostat mesilate respectively when their mean arterial pressure declined to 50 mmHg. The serum concentration of hydroxytryptamine (5-HT), prothrombin time (PT), and activated partial thromboplastin time (APTT) were determined as indicators of platelet activity and blood coagulation. In group-C, serum 5-HT was elevated significantly at 60 min after hemorrhagic shock but not so in group-E. The APTT at 30 and 60 min was shorter in group-C than in group-E. The PT at 30 min was also shorter in group-C. Plasma fibrin degradation products (FDP) increased at 60 min after the induction of shock in group-C. The results indicate that inadequate tissue perfusion in shock stimulates blood coagulation and that nafamostat mesilate might be beneficial in decreasing excessive blood coagulation.

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